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Clinical efficacy and drug resistance of anti-epidermal growth factor receptor (EGFR) therapy in colorectal cancer Minireview

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## Abstract

Colorectal cancer (CRC) ranked third in cancer related death and its incidence has been increasing worldwide. In recent decades important therapeutic advances have been developed in treatment of metastatic CRC (mCRC), such as <sup>16</sup>monoclonal antibodies against epidermal growth factor receptor (anti-EGFR), which provided additional clinical benefits in mCRC. However, anti-EGFR therapies have limited usage due to approximately 95% of patients with KRAS mutated mCRC <sup>2</sup>do not response to anti-EGFR <sup>11</sup>treatment. Thus, KRAS mutation is predictive of nonresponse to anti-EGFR therapies but it alone is not a sufficient basis to decide who should not be received such therapies because; approximately fifty percent (40-60%) of CRC patients with wild-type (WT) KRAS mutation also have poor response to anti-EGFR based treatment. This fact leads us to suspect that there must be other <sup>24</sup>molecular determinants of response to anti-EGFR therapies which have <sup>1</sup>not been identified yet. Current article summarizes the clinical efficacy of anti-EGFR therapies and also evaluates its resistance mechanisms.

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